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Childhood Lead Poisoning from Paint Chips: a Continuing Problem

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ABSTRACT *Although lead poisoning (*plumbism*) has been recognized for centuries, lead exposures still occur frequently today because of its varied uses and persistence in the environment. Despite the awareness of the adverse effects of lead on adults, childhood plumbism was first reported only about a century ago. Young children are one of the most vulnerable groups to the adverse effects of lead because of their rapidly developing central nervous systems. Federal regulations in the 1970s have been successfully implemented to decrease the amount of environmental lead by decreasing the content of lead in gasoline and indoor paint. However, almost 30 years after these laws were passed, inner-city housing with leaded paint still exists. We describe three children living in New York City who developed plumbism from the ingestion of leaded paint chips.*

INTRODUCTION

Lead was one of the first metals extensively utilized in early civilizations because of its abundance in the earth's crust, ease of refinement, and significant malleability. Although lead poisoning (*plumbism*) was first recognized centuries ago, lead exposures still occur frequently because of its varied uses and persistence in the environment.[1] Childhood plumbism was described as early as 1897 and subsequently has been linked since 1943 to deleterious effects on the cognitive development of children.[2,3] At that time, indoor house paint and leaded gasoline were major sources of poisoning. In urban environments, leaded paint continues to be a major source of exposure.[4–6] Despite an immensely successful public health initiative in the 1970s that focused on decreasing the environmental lead and implementation of widespread blood lead screening as part of routine health supervision for children, childhood lead poisoning has not been completely eradicated.[7] A major source may be older inner-city housing with peeling paint.[7]

Lead has adverse effects on multiple organ systems, especially the developing central nervous system.[8] The long-term neurologic toxicity of lead may manifest as subtle cognitive deficits, behavior changes, or decreased performance on psychometric intelligence scores (IQ testing).[9–13] Fatalities from the ingestion of lead paint are infrequently reported, but two cases in the past 12 years are well documented.[7,14] Children living in substandard housing are a potentially vulnerable group because of comorbidities that may include poor nutrition and limited access to health care.

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We describe three children living in New York City who developed lead poisoning from the ingestion of paint chips.

CASE 1

A 2-year-old boy with a history of ingesting peeling paint chips presented to his pediatrician for a routine evaluation and was found to have a venous blood lead level (BLL) of 60 μg/dL (normal <10 μg/dL). He was referred to the emergency department (ED) for further evaluation. The child had no significant past medical history and appeared developmentally normal. On physical examination, he was awake, alert, and nontoxic appearing. His laboratory findings are included in Table 1. Although he had an abdominal radiograph that did not reveal the presence of lead, radiographs of the extremities revealed an increased density of the distal femur consistent with a lead line (Fig. 1). He was treated with intramuscular edetate disodium calcium ($CaNa_2EDTA$) (1000 mg/m^2/day) for a total of 5 days. His hospital course was uneventful, and he was discharged to lead-safe housing. His venous blood lead level at discharge was 38 μg/dL; over the next 6 weeks, the level decreased to 26 μg/dL and then increased again to 34 μg/dL. Inspection by the New York City Department of Health Lead Bureau revealed multiple violations in a house built in 1931. Lead abatement was subsequently performed. The child continues to do well.

CASES 2 AND 3

Two 17-month-old twin brothers presented for their regular clinic appointments, and one was noted to have an elevated capillary lead level of 61 μg/dL. They were sent to the ED for evaluation. Both children were born at 32-weeks gestation, appeared developmentally normal, and were without any evidence of acute illness. The father reported that he saw both children with paint chips in their mouth within the past week. On physical examination, the children appeared awake, alert, and not acutely ill. Venous BLLs were found to be 52 μg/dL and 62 μg/dL, respectively; their other laboratory findings are included in Table 1. Abdominal radiographs of both children revealed several radiopaque foreign bodies in the small and large bowels; these foreign bodies were presumed to be leaded paint chips (Figs. 2 and 3). Long-bone radiographs revealed questionable increased density at the epiphyseal plates (Figs. 4 and 5).

The patients were both treated with whole bowel irrigation using polyethylene glycol electrolyte solution, and after repeat abdominal radiographs were negative,

TABLE 1. Laboratory values

Patient	Hemoglobin, g/dL	Hematocrit, %	Mean corpuscular volume, MCV	Peripheral smear red blood cell morphology	Abdominal radiograph	Long-bone radiographs	Whole blood lead level, μg/dL
1	10.4	31	67	N/A	Negative	+ Lead lines	56
2	13.3	39.3	77.0	Normal	Positive	? Lead lines	52
3	12.8	37.0	79.1	Normal	Positive	? Lead lines	62

N/A, not applicable.

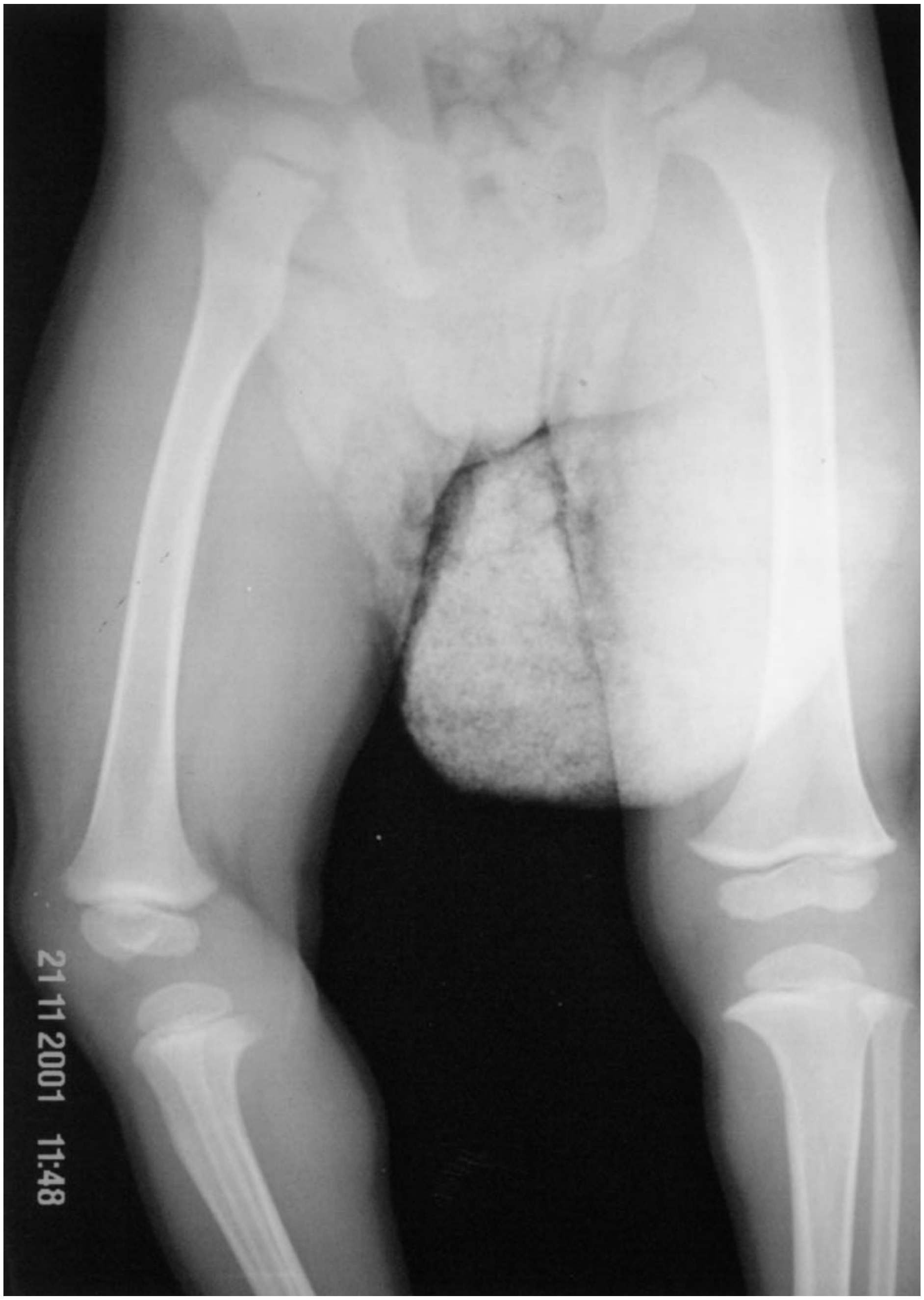

FIGURE 1. Long-bone radiograph demonstrating lead line at distal left femur of patient 1.

succimer (DMSA) was administered at an initial total daily dose of 30 mg/kg given orally in divided doses every 8 hours for the first 5 days, then the daily dose was 20 mg/kg divided every 12 hours for the next 14 days. They both remained asymptomatic during their hospital stay and were discharged to lead-safe housing. Repeat venous lead levels of both children while on chelation during their 5-day hospital-

FIGURE 2. Abdominal radiograph demonstrating several radiopaque densities consistent with lead chips in patient 2.

ization were 34 and 18 μg/dL for the first child and 43 and 21 μg/dL for the second child. Follow-up outpatient lead levels could not be obtained.

Inspection by the New York City Department of Health Lead Bureau revealed multiple violations in a house owned by the grandparents and built in 1931. Subsequent lead abatement was required, but no further follow-up records were avail-

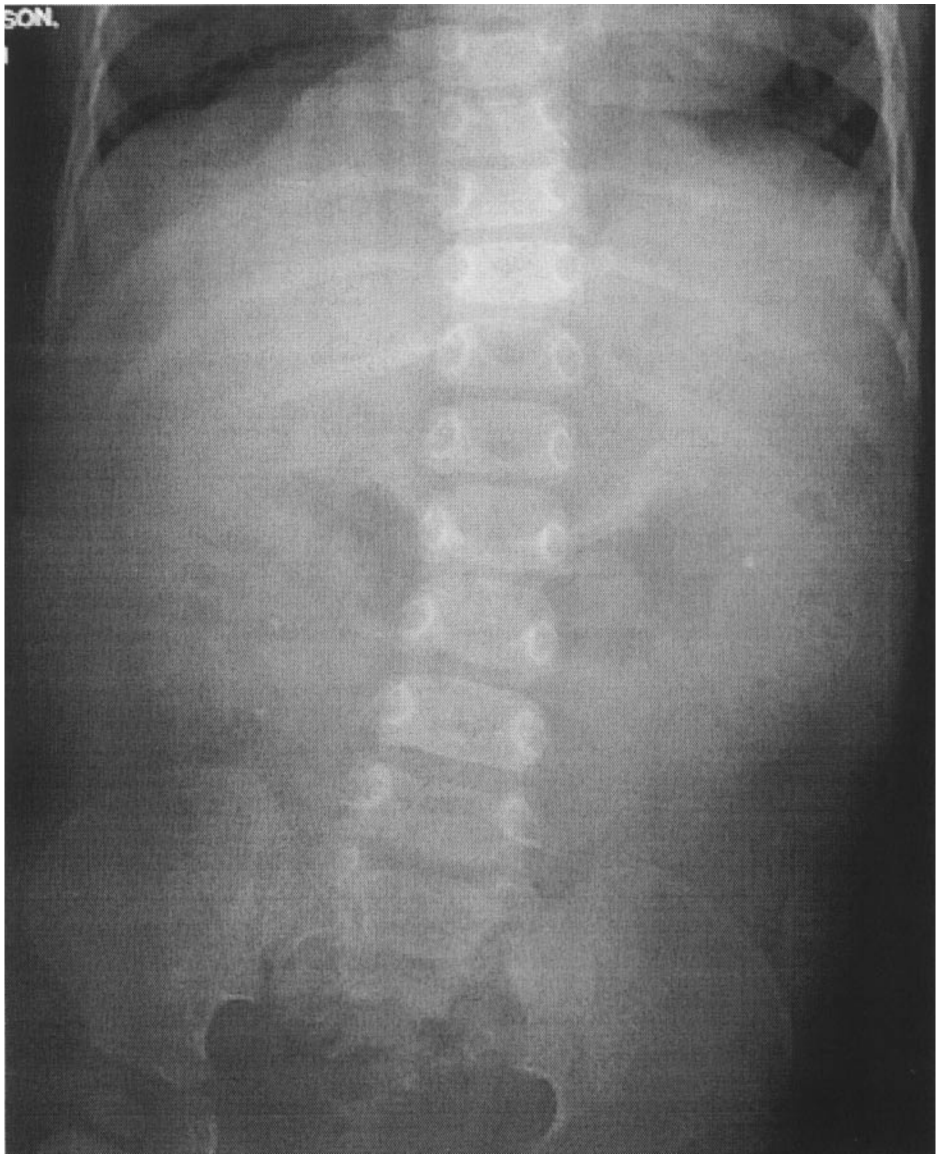

FIGURE 3. Abdominal radiograph demonstrating several radiopaque densities consistent with lead chips in patient 3.

able. The children were briefly visiting the grandparents' home, and their permanent address was located in Pennsylvania and deemed lead free.

DISCUSSION

Lead is a metal that possesses the versatile properties of malleability, a low melting point, and extreme stability in the environment. Products that may contain lead include pipes, solder, brass fixtures, ceramics, crystal, electric cable, paint, radiation shielding, gasoline, batteries, folk remedies, and cosmetics.[8] Prior to 1980, the three

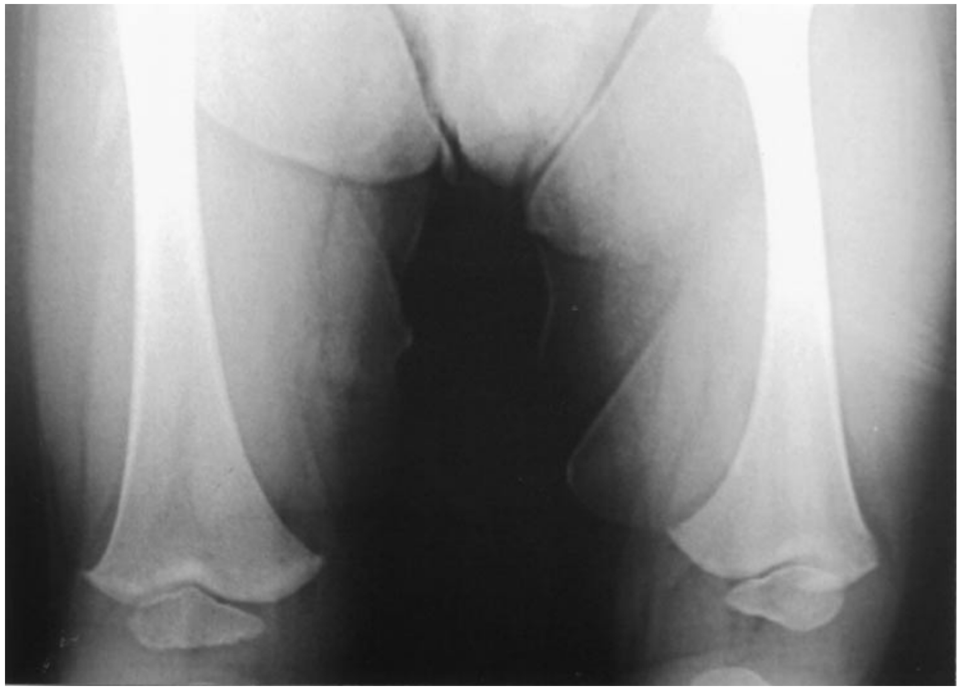

FIGURE 4. Long-bone radiograph demonstrating possible lead line at distal right femur of patient 4.

products that accounted for the majority of childhood lead exposures included lead-based indoor house paint, tetraethyl lead as a gasoline additive, and lead solder for sealing canned food.[15] The use of these products has been reduced markedly as a result of federal regulations limiting lead content and the development of safer alternatives. As a result, there has been an overall decline in mean blood lead levels because of these initiatives. Despite these regulations and mandatory screening guidelines, childhood lead poisoning remains a persistent health problem with potentially severe adverse sequelae involving multiple organ systems.

Lead may enter the body through various routes, with ingestion being the most common pathway in children.[1] Although inorganic lead salts are relatively insoluble in aqueous solutions such as gastrointestinal fluid, lead may be absorbed through similar mechanisms as those used for the absorption of essential elements (e.g., iron, calcium).[16] Hence, nutritional deficiencies may increase the absorption of lead and thereby increase the likelihood of toxicity.[16]

In the body, lead enters the intravascular space and attaches rapidly to red blood cells such that very little remains in the plasma.[17] Based on radioisotope studies, the half-life of lead in the blood of adults is about 3 weeks, with data for children less clear.[18] Lead is excreted by the kidneys or in feces via bile secretion.[17] The lead that remains in the body accumulates mostly in three compartments: blood, soft tissue, and bone.[17]

Physiologic Effects

Because lead enters most cells, multiorgan toxicity is expected. On a cellular level, lead is particularly attracted to proteins (including those with sulfhydryl, amine,

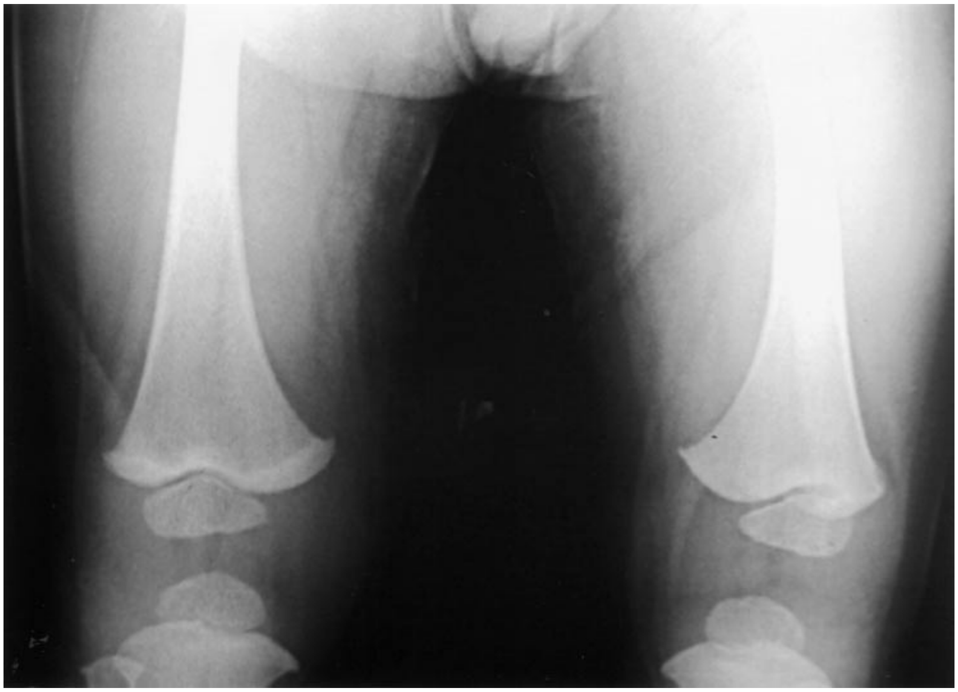

FIGURE 5. Long-bone radiograph demonstrating possible lead line at distal right femur of patient 5.

phosphate, and carboxyl groups).[17] In addition, because of valence similarities to calcium, lead may alter calcium-dependent processes.[17] Consequently, lead has major effects on the central and peripheral nervous systems, kidneys, and red blood cells and the metabolism of vitamin D and calcium.[17]

Lead induces toxicity in the red blood cell because it inhibits several enzymes (deltaaminoleulinic acid dehydratase, ferrochetalase, coproporphyrinogen decarboxylase) in the heme synthesis pathway.[19] Lead may result in hypertension by altering calcium-activated channels in the contractility of smooth muscle cells and cause subsequent increased activity of the Na^{+}-Ca^{+} exchange pump.[20] Other renal effects may occur as well by the impairment of mitochondrial function.[20] Last, neuronal toxicity can occur by multiple mechanisms. Disruption of the cerebral microvasculature results in injury to the blood-brain barrier and subsequent leakage of proteinaceous fluid.[21] The resultant increase in intracranial pressure may cause seizures, coma, and neuronal damage.[21] Peripheral neuronal damage may occur via demyelination and axonal degeneration.[22] A summary of the effects of lead on the various organ systems is described in Table 2.

Clinical Evaluation

Clinically, lead poisoning may be divided into acute and chronic toxicity. Fewer than 5% of children with lead poisoning are found to have lead poisoning solely based on their clinical presentation.[1] Gastrointestinal-related symptoms may be prominent in acute toxicity and include anorexia, nausea, vomiting, abdominal pain, and constipation. The BLL threshold for acute gastrointestinal symptoms is gener-

TABLE 2. Effects of lead toxicity on various organ systems

Organ system	Toxicity
Neurologic	*Children:* Acute exposures to high levels may produce encephalopathy. Low levels cause other neuropsychologic deficits. *Adults:* Lead encephalopathy may also occur (usually at higher levels); slowed nerve conduction and extensor muscle weakness are possible signs in persons chronically exposed (occupationally) to high lead levels.
Gastrointestinal	Abdominal pain, anorexia, vomiting, and constipation are common. Hepatitis and pancreatitis are rare; blue gingival lead line may develop (especially in adults).
Renal	Acute high-dose lead may cause proximal tubular dysfunction (Fanconi-like syndrome); continued repetitive exposures may lead to an irreversible lead nephropathy (interstitial nephritis). May contribute to the onset and development of hypertension, especially with higher exposures (primarily occupational). Destruction of erythropoietin may contribute to anemia.
Hematologic	Lead chronically interferes with hemoglobin synthesis by inhibiting multiple enzymes (δ-aminolevulinic acid dehydratase, ferrochelatase, etc.), resulting in a microcytic anemia. Lead may also produce a hemolytic anemia with acute massive exposures.
Endocrine	Lead prevents vitamin D conversion into its active form, 1,25-dihydroxyvitamin D, leading to impaired cell growth, maturation, and tooth and bone development. Usually occurs in children only with high BLLs (>62 μg/dL) and nutritional deficiency.
Cardiovascular	Myocarditis and cardiac dysfunction are rarely reported.
Skeletal	Bone formation and bone resorption may be impaired; bone growth and shortened stature may thus occur.
Reproductive and developmental	Ongoing occupational exposures may decrease sperm count and increase frequency of abnormal sperm, but studies on fertility are inconclusive. An association between higher lead levels and adverse pregnancy outcomes has been made, but this is probably less significant at current lower environmental lead levels. Prenatal exposure to low lead levels may increase the likelihood of prematurity and decreased birth weight.
Carcinogenic	Current data are inconclusive on carcinogenicity of lead in humans.

Sources: From Refs. 8 and 24.

ally accepted to be approximately 2.4 μmol/L (50 μg/dL). Another report[17] suggests that gastrointestinal symptoms rarely occur at BLLs of less than 0.97 to 2.2 μmol/L (20 to 45 μg/dL) and occur in the majority of patients with BLLs higher than 45 μg/dL. At levels above 4.83 μmol/L (100 μg/dL), encephalopathy is possible and is manifested by a change in mental status, ataxia, seizures, and coma.[1,9] Chronic sequelae include mental retardation, cranial and peripheral nerve palsies, and growth failure.[1]

Although most children with chronic low-level toxicity will be asymptomatic, the delayed effects on the developing brain are clearly demonstrated. In a study in which the mean length of patient follow-up was 11 years, elevated blood lead levels during childhood were associated with a significant decrease in academic performance in adolescence.[9] Several other studies also demonstrate an inverse correlation of BLL to IQ; in general, about one quarter to one half of an "IQ point" is lost for each 0.04826 μmol/L (1 μg/dL) increase in BLL over serial IQ measurements during preschool years.[10–13] It is this decrease in cognitive function that has provided the major impetus for current public health efforts.

Laboratory Evaluation

Aside from an elevated BLL, laboratory values in acute lead exposures will generally be normal. The Centers for Disease Control and Prevention (CDC)[23] currently considers children to have an elevated BLL if it is above 10 μg/dL. In chronic toxicity, several laboratory indices may indicate severity and/or duration of toxicity. Measurement of increased quantities of heme precursors such as free erythrocyte protophorphyrin (FEP) level reflects impaired heme synthesis. The EP assay is considered normal below 35 μg/dL and, because of the lag time after exposure to lead, is not useful in acute lead exposures.[24] As a consequence of bone marrow toxicity in children, a hypochromic, microcytic anemia may be seen on the complete blood count (CBC) and basophilic stippling in red blood cells visualized on the peripheral blood smear.[25]

Radiodensities indicating growth arrest may be apparent on radiographs of weight-bearing surfaces of long bones. Abdominal radiographs are only useful to detect a recent ingestion of leaded paint chips or other lead-containing foreign bodies, which must be of sufficient size to be visualized.[26] In one study conducted in a lead poisoning clinic, children with elevated BLLs rarely had positive abdominal radiographs.[26] It is therefore assumed that repeated (i.e., chronic) ingestion of paint chips can be detected if the timing of the radiograph is coincidental. Other radiologic tests, such as computed tomography (CT) and magnetic resonance imaging (MRI) studies of the brain, may reveal signs of increased intracranial pressure and, rarely, cerebellar calcifications or optic neuritis.[27–29]

As discussed above, multiple organ systems may be affected. For example, the kidneys may be damaged, resulting in an interstitial nephritis, resulting in abnormalities of renal indices (i.e., serum blood urea nitrogen and creatinine).[24] Lead can also rarely cause a Fanconi syndrome with tubular dysfunction (i.e., glycosuria, proteinuria, and phosphaturia).[24]

Management

Management of lead poisoning due to the ingestion of leaded paint chips is dependent on whether it is acute or chronic. If radiographs of the abdomen reveal opacifications consistent with paint chips, gastrointestinal decontamination via whole bowel irrigation using polyethylene glycol electrolyte lavage solution is probably warranted. Whole bowel irrigation should be performed until the abdominal radiographs are clear.

After a child is diagnosed, based on clinical or laboratory parameters, with lead poisoning and has received adequate gastrointestinal decontamination, chelation may be required. (A detailed discussion of chelation therapy is beyond the scope of this discussion, but the reader is referred to the CDC recommendations.[23]) Chelation may effectively reduce the body lead burden and can be performed with multiple agents, including $CaNa_2EDTA$, dimercaprol (BAL), D-penicillamine, and succimer (DMSA). Because these drugs have different risk : benefit profiles, varied routes of administration, and different protocols, physicians with experience in chelation therapy should be consulted prior to their implementation.

In general, oral chelation agents (DMSA, penicillamine) are used for asymptomatic children with moderately elevated BLLs (45–69 μg/dL). In children with higher BLLs or symptoms of lead toxicity, parenteral agents ($CaNa_2EDTA$, BAL) are usually indicated individually or in combination. Although a recent prospective study of asymptomatic children with BLLs between 20 and 44 μg/dL shows no difference in neurologic outcome in children treated with DMSA compared to untreated controls,[30] controversy still exists regarding treatment for this group.[31,32] The evidence that chronic lead exposure results in neurologic impairment, however, compels us to recommend chelation therapy with the relatively benign chelating agent DMSA for many children with BLLs less than 45 μg/dL. Despite the efficacy of DMSA in decreasing the body's total lead burden, the end point of therapy still remains a separate and unanswered issue.

The primary goal to limit lead-induced neurologic and developmental impairment in children is prevention.[33] Once lead has entered the body, especially the skeleton, it is very difficult to remove. For the majority of children, the success of treatment depends most heavily on identifying and eliminating the sources of lead exposure. Attempted modifications of children with hand-to-mouth and pica behavior may also be necessary. In addition, if significant quantities of lead are found in the home, lead abatement should be performed.

CONCLUSION

It is clear that, although the public health initiatives of the last few decades have substantially reduced the overall amount of lead in the environment, lead poisoning may still result in children from the ingestion of lead-based paint chips. Housing conditions in which leaded paint is still present must be properly abated to prevent ongoing exposures. In addition, because low-level lead poisoning may be difficult to detect clinically, routine screening will help detect significant exposures long before overt clinical manifestations occur. Children with abnormal behavior (such as pica), siblings of known cases, and children with other potential risk factors should be routinely evaluated for possible lead poisoning. Prevention is clearly of the utmost importance, but prompt diagnosis and subsequent treatment of lead poisoning should help minimize possible adverse long-term sequelae.

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